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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/519,665	03/06/2000	Steve H Hinrichs	N1374-007	8712
110	7590	04/14/2004	EXAMINER	
DANN, DORFMAN, HERRELL & SKILLMAN 1601 MARKET STREET SUITE 2400 PHILADELPHIA, PA 19103-2307			DAVIS, MINH TAM B	
		ART UNIT		PAPER NUMBER
		1642		

DATE MAILED: 04/14/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/519,665	HINRICHES, STEVE H	
	Examiner	Art Unit	
	MINH-TAM DAVIS	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
 - If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
 - If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
 - Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) Responsive to communication(s) filed on 30 January 2004.
- 2a) This action is **FINAL**. 2b) This action is non-final.
- 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) Claim(s) 1-4, 17-20 and 56-64 is/are pending in the application.
- 4a) Of the above claim(s) 63 and 64 is/are withdrawn from consideration.
- 5) Claim(s) _____ is/are allowed.
- 6) Claim(s) 1-4, 17-20 and 56-62 is/are rejected.
- 7) Claim(s) _____ is/are objected to.
- 8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) The specification is objected to by the Examiner.
- 10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) All b) Some * c) None of:
1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) Notice of References Cited (PTO-892)
- 2) Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date _____.
- 4) Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____.
- 5) Notice of Informal Patent Application (PTO-152)
- 6) Other: _____.

DETAILED ACTION

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Applicant cancels claims 8-14.

Applicant adds new claims 56-64.

Since applicant has elected group IV, drawn to a method for modulating transcription factor-mediated gene expression, comprising exposing said transcription factor to an inhibitory polypeptide agent polypeptide which binds to a linker domain of said transcriptional factor, wherein said transcription factor is EWS/ATF1, and wherein said linker domain is located adjacent to the DNA binding domain of said transcriptional factor, for action on the merits for the originally presented invention, this invention has been constructively elected by original presentation for prosecution on the merits.

Accordingly, the embodiments of new claims 63-64, directed to a method for modulating transcription factor-mediated gene expression", comprising exposing said transcription factor to an inhibitory agent which binds to a linker domain of said transcriptional factor, wherein said transcription factor is EWS/ATF1, wherein said linker domain is located adjacent to the DNA binding domain of said transcriptional factor, wherein said inhibitory agent is expressed intracellularly, and wherein said inhibitory agent is encoded for in a retroviral vector, have been withdrawn from consideration as being directed to a non-elected invention. See 37 C.F.R. 1.142(b) and M.P.E.P. 821.03. Newly submitted claims 63-64 are directed to an invention that is independent or distinct from the invention originally claimed for the following reasons:

The invention of new claims 63-64 is drawn to gene therapy, using a retroviral vector expressing an inhibitory agent, which is different from the originally presented invention, using an inhibitory polypeptide agent, an antibody, that binds to a linker domain of a transcriptional factor EWS/ATF1. The above two methods are materially distinct methods, which differ at least in objectives, method steps, reagents and/or dosages and/or schedules used, response variables, and criteria for success.

Accordingly, claims 1-4, 17-20, 56-62, the transcriptional factor EWS/ATF1, its linker domain of species residues 205-219 of the ATF1 of SEQ ID NO:1, species antibody or subcomponent of an antibody and species sarcoma are examined in the instant application, are being examined. Claims 63-64 are withdrawn from consideration as being drawn to non-elected invention.

It is noted that the issues concerning the generic language "any inhibitory agent" and "any linker domain" are presently not discussed, because of the species election of an antibody as an inhibitory agent, and linker domain of species residues 205-219 of the ATF1 of SEQ ID NO:1.

The following are the remaining rejections.

OBJECTION

New claim 61 is objected to for the use of the language "sFv4". Does Applicant mean scFv4?

**REJECTION UNDER 35 USC 112, FIRST PARAGRAPH, WRITTEN DESCRIPTION,
NEW REJECTION**

Claims 1-4, 7-12, and new claims 56-62 are rejected for lack of a clear written description of a method for modulating “transcription factor-mediated gene expression”.

Claims 1-4, 7-12, 56-62 are drawn to a method for “modulating EWS/ATF1-mediated gene expression”, comprising exposing said transcription factor to an inhibitory agent which binds to a linker domain of said transcriptional factor, wherein said transcription factor is EWS/ATF1, and wherein said linker domain is located adjacent to the DNA binding domain of said transcriptional factor. Said modulation occurs within a cancerous cell, or a sarcoma or mesenchymal sarcoma, or a clear cell sarcoma.

It is noted that a method for modulating “transcription factor EWS/ATF1-mediated gene expression” encompasses a method for modulating expression of a genus of genes with unknown structure, the expression of which is mediated by EWS/ATF1.

Although drawn to DNA arts, the findings in University of California v. Eli Lilly and Co., 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997) and Enzo Biochem, Inc. V. Gen-Probe Inc. are relevant to the instant claims. The Federal Circuit addressed the application of the written description requirement to DNA-related inventions in University of California v. Eli Lilly and Co., 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997). The court stated that [a] written description of an invention involving a chemical genus, like a description of a chemical species, requires a precise definition, such as by structure,

formula, [or] chemical name, of the claimed subject matter sufficient to distinguish it from other materials. Id. At 1567, 43 USPQ2d at 1405. The court also stated that

a generic statement such as vertebrate insulin cDNA or mammalian insulin cDNA without more, is not an adequate written description of the genus because it does not distinguish the genus from others, except by function. It does not specifically define any of the genes that fall within its definition. It does not define any structural features commonly possessed by members of the genus that distinguish them from others. One skilled in the art therefore cannot, as one can do with a fully described genus, visualize or recognize the identity of the members of the genus. A definition by function, as we have previously indicated, does not suffice to define the genus because it is only an indication of what the gene does, rather than what it is.

Id. At 1568, 43 USPQ2d at 1406. The court concluded that naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material. Id.

Finally, the court addressed the manner by which a genus of cDNAs might be described. "A description of a genus of cDNAs may be achieved by means of a recitation of a representative number of cDNAs, defined by nucleotide sequence, falling within the scope of the genus or of a recitation of structural features common to the members of the genus, which features constitute a substantial portion of the genus."

The Federal Circuit has recently clarified that a DNA molecule can be adequately described without disclosing its complete structure. See Enzo Biochem, Inc. v. Gen-Probe Inc., 296 F.3d 1316, 63 USPQ2d 1609 (Fed. Cir. 2002). The Enzo court adopted the standard that “the written description requirement can be met by “show[ing] that an invention is complete by disclosure of sufficiently detailed, relevant identifying characteristicsi.e., complete or partial structure, other physical and/or chemical properties, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics.

Id. At 1324, 63 USPQ2d at 1613 (emphasis omitted, bracketed material in original).

The inventions at issue in Lilly and Enzo were DNA constructs per se, the holdings of those cases are also applicable to claims such as those at issue here. A disclosure that does not adequately describe a product itself logically cannot adequately describe a method of using that product.

Thus, the instant specification may provide an adequate written description of genes, the expression of which are mediated by transcription factor EWS/ATF1, per Lilly by structurally describing a representative number of genes that are mediated by EWS/ATF1, or by describing “structural features common to the members of the genus, which features constitute a substantial portion of the genus.” Alternatively, per Enzo, the specification can show that the claimed invention is complete “by disclosure of sufficiently detailed, relevant identifying characteristics, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics.”

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In this case, the specification does not describe genes, the expression of which are mediated by transcription factor EWS/ATF1, to practice the method of claims 1-4, 17-20, 56-62, in a manner that satisfies either the Lilly or Enzo standards. The specification does not provide the complete structure of any genes that are mediated by the transcription factor EWS/ATF1, nor does the specification provide any partial structure of such genes, nor any physical or chemical characteristics of genes that are mediated by the transcription factor EWS/ATF1, nor any functional characteristics coupled with a known or disclosed correlation between structure and function. Although the specification discloses the activation of CRE is inhibited by binding of scFv4 to EWS/ATF1, this does not provide a description of genes that are mediated by the transcription factor EWS/ATF1, that would satisfy the standard set out in Enzo.

The specification also fails to describe genes that are mediated by the transcription factor EWS/ATF1 by the test set out in Lilly. The specification describes only inhibition of CRE activation. Therefore, it necessarily fails to describe a representative number of such species. In addition, the specification also does not describe "structural features common to the members of the genus, which features constitute a substantial portion of the genus."

Thus, the specification does not provide an adequate written description of genes that are mediated by the transcription factor EWS/ATF1, that is required to practice the claimed invention. Since the specification fails to adequately describe the product that is modulated by the claimed method, it also fails to adequately describe the claimed method.

For the reasons set forth above, Applicant was not in possession of a method for modulating "transcription factor EWS/ATF1-mediated gene expression", comprising exposing said transcriptional factor to an inhibitory agent which binds to a linker domain of said transcriptional factor, wherein said linker domain is located adjacent to the DNA binding domain of said transcriptional factor.

REJECTION UNDER 35 USC 112, FIRST PARAGRAPH, SCOPE

Rejection under 35 USC 12, first paragraph of claims 1-4, 17-20, pertaining to lack of enablement for **in vivo method of modulating EWS-ATF1-mediated gene expression in cancer therapy as contemplated, or treating cancer as contemplated**, remains for reasons already of record in paper No.10. New claims 56-62 are rejected for the same reasons of record.

Applicant argues that the scFv4 has been demonstrated to be efficacious *in vivo* in Jean et al. (Oncogene (2000) 19:2721-30). Specifically, intracellular expression of an scFv4 fragment derived from mAb4 (page 2722, left column) in human melanoma cells in an *in vivo* model was determined to render the cells apoptotic and inhibit tumor growth and metastasis (page 2725, right column). Applicant asserts that while the scFv 4fragment employed was directed to inhibiting ATF-I transcriptional activity, a skilled artisan would readily appreciate that the scFv4 fragment would inhibit EWS/ATF1 mediated gene expression.

The recitation of Jean et al is acknowledged and entered.

Applicant's arguments set forth in paper of 01/30/04 have been considered but are not deemed to be persuasive for the following reasons:

A. It is noted that in the example by Jean et al, a melanoma cell line has been transfected with a vector expressing scFv4 before being injected into nude mice, and tumor growth of said transfected cell line in nude mice is subsequently monitored. Thus it is questionable that the example taught by Jean et al would represent an in vivo model for modulating EWS-ATF1-mediated gene expression, or treating cancer, using scFv4 peptide; e.g. the single chain antibody scFv4 would be administered into a treated subject via conventional methods, such as intravenously etc., One cannot extrapolate the teaching of Jean et al to the claims, because it is well known in the art that single chain antibody peptide is not stable in vivo, e.g., in circulation (Li Q et al, 1998, Cancer Immunol Immunotherapy, CII (Germany): 47(3): 121-30). Thus, one cannot predict that scFv4 would not be degraded or absorbed by fluids, cells and tissues where the scFv4 peptide has no effect, and could reach the targeted tumor cells at a sufficient concentration, for a sufficient period of time, for modulating EWS-ATF1-mediated gene expression in a conventional cancer therapy. Further, since mAb4 and its fragment, scFv4, bind to the transcriptional factor ATF1/CREB within the nucleus of the tumor cells expressing ATF1/CREB, one cannot predict that scFv4, or at least mAb4 could penetrate both the outer cellular membrane and the nucleus membrane to reach the target protein within the nucleus, where the activity of scFv4 or mAb4 is to be exerted.

For the reasons set forth above, and in view that cancer treatment is unpredictable, as taught by Gura, Jain, Curti and Hartwell et al, all of record, one cannot predict that the claimed method would be effective in in vivo modulating EWS-ATF1-mediated gene expression in cancer therapy as contemplated, or treating cancer as contemplated.

B. Moreover, claims 56-58 reads on a method for treating any cancers, or any sarcoma or any mesenchymal sarcoma.

The specification discloses that expression of scFv4 in a single tumor cell line, SU-CCS-1 endogenously expressing EWS/ATF1 fusion protein decreases the expression of CRE-luc reporter and leads to loss of cell viability and apoptosis (Examples 17-18, on pages 86-90). The specification further discloses that tumor cell line, SU-CCS-1 is highly reminiscent of Clear cell sarcoma.

One cannot extrapolate the teaching in the specification to the scope of the claims, because even if the claimed method could be used for in vivo treating SU-CCS-1 like cancer such as Clear cell sarcoma, one cannot predict that any cancer could be treated using the claimed method, because different cancers have different etiology and characteristics, and different responses to a drug, and because Applicant has not shown that any cancer cells, or any sarcoma or any mesenchymal sarcoma would express EWS/ATF1 fusion protein, which is the target of the inhibitory antibody of the claimed method.

REJECTION UNDER 35 USC 112, FIRST PARAGRAPH, SCOPE, NEW REJECTION

1. Claims 1-4, 7-12, 56-62 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method for inhibiting CRE activation of clear cell sarcoma expressing EWS/ATF1 in vitro, **does not reasonably provide enablement for a method for modulating the expression of any gene, the expression of which are mediated by EWS/ATF1.** The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

Claims 1-4, 7-12, 56-62 are drawn to a method for "modulating EWS/ATF1-mediated gene expression", comprising exposing said transcription factor to an inhibitory agent which binds to a linker domain of said transcriptional factor, wherein said transcription factor is EWS/ATF1, and wherein said linker domain is located adjacent to the DNA binding domain of said transcriptional factor. Said modulation occurs within a cancerous cell, or a sarcoma or mesenchymal sarcoma, or a clear cell sarcoma.

Claims 1-4, 7-12, 56-62 encompass a method for "modulating expression of a whole universe of unknown genes which could be mediated by the transcriptional factor EWS/ATF1", comprising exposing said transcription factor to an inhibitory agent which binds to a linker domain of said transcriptional factor, wherein said linker domain is located adjacent to the DNA binding domain of said transcriptional factor.

The specification discloses that it has been determined that amino acids 205-219 of ATF1 protein of SEQ ID NO:1 is a linker domain for ATF1 (p.12), wherein a linker domain is a unique sequence for transcriptional factors, such as the linker domains of

ATF1, CREB and GCN4 (members of the b-ZIP family), that has been determined to be the epitope of mAb41.4, and wherein the linker domain links the transcription activation domain (TAD) to the DNA binding domain and optionally dimerization region (p. 14).

The specification discloses that antibody mAb4 inhibits transcription using a murine proliferating cell nuclear antigen gene promoter (PCNA) as template (p.52), and that expression of scFv4 in Hela cells transfected with pEWS/ATF1 decreases the reporter CRE-luc expression, suggesting that scFv4 is capable of inhibiting CRE activation by EWS/ATF1 in Hela cells, and consequently inhibiting transcription of genes (Example 17 on pages 85-86). The specification also discloses that expression of scFv4 in tumor cell line SU-CCS-1 endogenously expressing EWS/ATF1 fusion protein decreases the expression of CRE-luc reporter, and leads to loss of viability and apoptosis (Examples 17-18, on pages 86-90). Moreover, the specification discloses that although full length mAb4 does not inhibit CREB binding to DNA, scFv4 inhibits both ATF-1 and CREB binding to DNA (Examples 9-10 on pages 58-61).

It is noted that transcriptional factors such as ATF1 and CREB and CREM of the bZip transcriptional factors subfamily regulate transcription through binding to cyclic AMP response element (CRE) (Bosilevac, JM et al, 1999, of record, p.34811, second column, paragraph before last).

There is no disclosure of which genes, the expression of which are mediated by the inhibiting action of scFv4 on CRE activation by EWS/ATF1.

One cannot extrapolate the teaching in the specification to the scope of the claims. It is not clear which genes would be mediated by EWS/ATF1 and modulated by

the claimed method, because it is well known in the art that not any genes would be modulated by a transcriptional factor, and because one cannot predict that any genes would be affected by inhibition of CRE activation by EWS/ATF1.

Further, modulating a gene expression encompasses increasing or decreasing expression of a gene. The specification only discloses that scFv4 decreases the reporter CRE-luc expression, suggesting that scFv4 is capable of inhibiting CRE activation by EWS/ATF1 in Hela cells. There is no indication that scFv4 would also increase gene expression, by inhibiting CRE activation, nor is there any correlation between inhibition of CRE activation by sFv4 and increasing gene expression. As disclosed in the specification, the region that is involved in transcriptional activation is the amino terminal region of ATF1 bound by mAb1, which is different from the carboxy-terminal half of ATF1 where the epitope of scFv4 is (p.52-54).

In the absence of an objective evidence of specific genes that are modulated by the claimed method, in view of the complex nature of the claimed invention, and little is known in the art about the claimed invention, one of skill in the art would be forced into undue experimentation to practice the claimed invention.

REJECTION UNDER 35 USC 102(a)

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

Claims 1-4, 17-20 remain rejected under 35 U.S.C. 102(a) as being anticipated by Bosilevac, JM et al, 1999, JBC, 274(49): 34811-8, for reasons already of record in paper No:10. New claims 56-62 are rejected for the same reasons already of record.

Applicant asserts that the instant inventor, Steven H. Hinrichs, is also a co-author of Bosilavac et al. Applicant asserts that it is logically self-evident that the publication relating to use of an antibody fragment to inhibit EWS/ATFI binding to target DNA and related method steps in Bosilevac et al. could not have occurred before the invention thereof by Steven Hinrichs. Applicant asserts that furthermore, Bosilevac et al. is not citable as prior art in the present case, as the law is well settled that one's own work is not prior art under 5102(a) even though it has been disclosed to the public in a manner or form which otherwise would fall under 5102(a). In re Katz, 215 U.S.P.Q. 14 (CCPA 1982). Applicant asserts that consequently, Bosilevac et al. also can not support a rejection under 35 U.S.C. 5103. Ex Parte Oetiker, 23 U.S.P.Q.Zd 1641 (BPAI 1992).

Applicant's arguments set forth in paper of 01/30/04 have been considered but are not deemed to be persuasive for the following reasons:

It is noted that Steven Hinrichs is the sole inventor of the instant application. The reference by Bosilevac et al however contains several authors besides Steven Hinrichs. Thus the teaching of Bosilevac et al is work by another inventive entity.

REJECTION UNDER 35 USC 103

Claims 1-4, 17-20 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Orten et al, 1994, JBC, 269(51): 32254-33263, or Bosilevac, JM et al, supra, in view of Brown et al, 1995, Oncogene, 10: 1749-1756, for reasons already of record in paper No:10. New claims 56-62 are rejected for the same reasons already of record.

Applicant argues that the Examiner indicates that "the structure of different transcription factors and their putative linker domains are however different and are not necessarily exposed such that an antibody fragment scFv could bind to" the linker domain. Applicant asserts that moreover, the Examiner notes at page 16 of the Official Action that "the context of the protein [is] important and affect(s) the antibody binding affinity." Applicant asserts that indeed, Bosilevac et al. teach that "the contribution of EWS to the overall conformation of the chimeric protein is unknown" and thus it is also unknown "whether the addition of the EWS domain would block the epitope of mAb4" (page 34814, left column). Applicant asserts that in as much as the fusion protein EWS/ATFI is twice the size of the ATFI transcription factor (531 amino acids to 271 amino acids, see page 34813 of Bosilevac et al.), a skilled artisan would readily appreciate that the epitope of mAb4 could be obscured to antibody or antibody fragment entry and binding by the large EWS protein fused to ATFI.

Applicant concludes that based on the Examiner's own reasoning that linker domain of different transcription factors are not necessarily exposed to antibody binding

and that the effects of the EWS fusion on the structure and availability of certain epitopes of ATF1 are completely unknown, a skilled artisan would be precluded from reasonably expecting mAb4 to be able to bind EWS/ATF1 and inhibit DNA binding in the same way mAb4 inhibits DNA binding by ATF1.

Applicant's arguments set forth in paper of 01/30/04 have been considered but are not deemed to be persuasive for the following reasons:

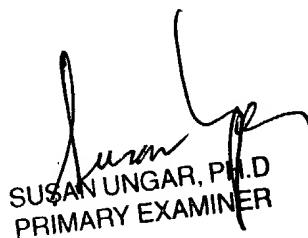
Contrary to Applicant's assertion, Bosilevac et al teach that the C-terminal of EWS/ATF1 retains the mAb4 epitope and that this epitope is accessible to binding to scFv4 (p.34812, first column, lines 19-21).

Further, one would have expected that EWS does not block the epitope of scFv4, which is within the ATF1 bZip domain. EWS/ATF1 is predicted to bind to ATF1 binding sites on the promoters via the ATF1 bZIP domain as taught by Brown et al (Brown et al, p.1749, second column, third paragraphs). Based on the teaching of Brown et al, one would have expected that the ATF1 bZIP domain of EWS/ATF1 is accessible, and is not blocked by EWS, because otherwise, EWS/ATF1 would not bind to ATF1 binding sites on the promoters via the ATF1 bZIP domain.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to MINH-TAM DAVIS whose telephone number is 571-272-0830. The examiner can normally be reached on 9:30AM-4:00PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, YVONNE EYLER can be reached on 571-272-0871. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).



SUSAN UNGAR, PH.D.
PRIMARY EXAMINER

MINH TAM DAVIS

March 30, 2004